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Acinetobacter baumannii clonal lineages I and II harboring different carbapenem-hydrolyzing-β-lactamase genes are widespread among hospitalized burn patients in Tehran

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Summary The aim of this study was to analyze antimicrobial resistance patterns and their encoding genes and genotypic diversity of *Acinetobacter baumannii* isolated from burn patients in Tehran, Iran. The presence of extended-spectrum beta-lactamase- and bla_{OXA}-encoding genes among 37 multidrug resistant (MDR) *A. baumannii* strains isolated from patients hospitalized in a teaching hospital in Tehran was evaluated. Susceptibility to 7 antibiotics was tested by disk agar diffusion and to polymyxin B and colistin was tested by E-test, according to CLSI guidelines. All isolates were then analyzed by PCR for the presence of bla_{IMP}, bla_{VIM}, bla_{SIM} $bla_{OXA\text{-}23}$,

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bla_{OXA-24}, and bla_{OXA-58}-like carbapenemase genes, and bla_{OXA-51}-like, bla_{TEM}, bla_{SHV}, bla_{PER}, bla_{VEB}, and bla_{GIM} genes. Genotyping of *A. baumannii* strains was performed by repetitive sequence-based (REP)-PCR and cluster analysis of REP-PCR profiles. *A. baumannii* isolates were assigned to international clones by multiplex PCR sequence group analysis. Twenty-five *A. baumannii* isolates were classified as MDR, and 12 were classified as extensively drug resistant. All isolates were susceptible to colistin and polymyxin B. Eighty-one percent of the isolates was resistant to imipenem or meropenem and harbored at least one or both of the bla_{OXA-23}-like or bla_{OXA-24}-like carbapenemase genes. Co-existence of different resistance genes was found among carbapenem-resistant isolates. Multiplex PCR sequence group analysis most commonly assigned *A. baumannii* isolates to international clones I (18/37; 48.6%) and II (18/37; 48.6%). An alarming increase in resistance to carbapenems and the spread of bla_{OXA-23}-like and/or bla_{OXA-24}-like carbapenemase genes was observed among *A. baumannii* strains belonging to clonal lineages I and II, isolated from burn patients in Tehran.

Introduction

Acinetobacter baumannii is a gram-negative bacterium causing hospital-acquired infections in critically ill patients [1]. The spread of a restricted number of clonal lineages that have been selected because of their multiple drug resistance has been reported worldwide [1—4].

Antimicrobial resistance owing to enzymatic degradation, modification of targets, and active efflux of drugs, together with persistence of the bacterium in contaminated environments are responsible for epidemics of *A. baumannii* in hospital settings [1,2,5,6]. Carbapenems are considered the first-line drugs for treatment of *A. baumannii* infections in Iran; however, resistance to these drugs is increasing owing to the overexpression of efflux pumps and to the ability of the bacterium to produce different carbapenemases, in particular class D carbapenemases [1,2,5,6]. *A. baumannii* is increasingly causing infections in burn units worldwide including Iran [7—9]. In addition, *A. baumannii* strains resistant to different classes of antibiotics including carbapenems have reportedly been isolated from burn patients in Iran [8,9]. The alarming increase in resistance to carbapenems in Iranian burn units from 2001 to 2009 [8,9] prompted us to analyze whether carbapenem resistance in *A. baumannii* strains isolated from burn patients was caused by horizontal gene transfer or selection of carbapenem-resistant epidemic clones, which has not been investigated to date. Previous studies in different clinical settings have shown the spread of *A. baumannii* isolates belonging to different repetitive sequence-based (REP)-PCR profiles and international clones I and II in Iran [10—12]. The aim of this study was to analyze the antimicrobial resistance patterns, β-lactamase gene content, genotypic diversity, and clonal lineage distribution of *A. baumannii* isolates in a burn ward of a university teaching hospital in Tehran, Iran.

Material and methods

Study population

In total, 37 non-replicate *A. baumannii* isolates associated with infection in patients admitted to the burn ward of a teaching hospital in Tehran were selected for this study. The Motahari Burn and Reconstruction Center is one of the few large, highly equipped, tertiary burn centers in Iran, providing care to severely burned patients from the province of Tehran and to those with complications that have been referred from other centers across the country. All patients were admitted immediately after burn injuries, except for referral cases.

Available epidemiological data from these isolates were retrospectively collected from patient charts and diagnostic microbiological laboratory culture reports. We used the criteria for diagnosis of infections in burn units described by Santucci and colleagues [13]. In addition, the following criteria were adopted to define wound infection or colonization: bacteria in the wound and wound eschar

at high concentration (>10^5 bacteria/g tissue) and no invasive infection was considered as infection; low concentration of bacteria in the wound (<10^5 bacteria/g tissue) and no invasive infection was considered as colonization [14].

All isolates were identified as *A. baumannii* by API 20NE and *gyr*B multiplex PCR, as previously described [15]. *A. baumannii* strains were considered multidrug resistant (MDR) and extensively drug resistant (XDR) if they were resistant to ≥1 agent in ≥3 antimicrobial categories and resistant to ≥1 agent in all but ≤2 categories, respectively, according to Magiorakos et al. [4].

Because we performed a retrospective study, no attempts were made to recover *Acinetobacter* spp. from the hospital environment.

Antimicrobial susceptibility testing

Susceptibility to imipenem (10 μg), meropenem (10 μg), gentamicin (10 μg), cefepime (30 μg), ciprofloxacin (5 μg), piperacillin—tazobactam (100/10 μg), and amoxicillin-sulbactam (10/10 μg) was evaluated by disk agar diffusion, and susceptibility to polymyxin B and colistin was evaluated by the E-test method (AB BIODISK, Solna, Sweden). Results were interpreted according to CLSI guidelines [16]. *Escherichia coli* ATCC 25922 and 35218 and *Pseudomonas aeruginosa* ATCC 27853 were used as controls.

Characterization of β-lactamase genes

All isolates were analyzed by PCR for detection of bla_{OXA}-like carbapenemase, extended-spectrum beta-lactamase, and metallo-β-lactamase, with the specific primers listed in Supplementary Table 1 [15,17—20]. Chromosomal DNA was extracted using a genomic DNA purification kit (Bioneer, Seoul, Korea). PCR was performed in a final volume of 25 μL containing 1× PCR buffer, 2.5 mm $MgCl_2$, 0.2 mM dNTP mix, 10 pmol of each primer, and 50 ng of template DNA. The amplification reactions were performed at 94 °C for 5 min, followed by 30 cycles of denaturation 30 s at 94 °C, 30 s of annealing (the different temperature of which are listed in Supplementary Table 1), and of extension 60 s at 72 °C. *A. baumannii* NCTC12156, NCTC13302, NCTC13303, and NCTC13304 were used as standard positive controls for bla_{OXA-51}, bla_{OXA-23}, bla_{OXA-24}, and bla_{OXA-58}-like carbapenemases, respectively. For all other PCR amplifications, the products obtained were considered positive based on amplicon size and direct sequencing of selected amplicons. In the case of negative PCR, in which a positive control was not used, PCR amplifications were repeated at least twice for these genes. A negative control was run with every PCR.

Supplementary Table 1 related to this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jiph.2015.04.030.

REP-PCR typing

All the isolates were analyzed by the REP-PCR typing method to find common REP-types among all isolates according to a previous report [21]. The primers used for REP-typing were F: 5′- IIIGCGCCGICATCAGGC-3′ and R: 5′-ACGTCTTATCAGGCCTAC- 3′. Cluster analysis of REP-PCR profiles was performed using GelCompar II v. 3.5 (Applied Maths, Sint-Martens-Latem, Belgium) with the unweighted pair-group method with arithmetic mean (UPGMA). The Dice correlation coefficient was used with a tolerance of 1.5% to analyze similarities between the banding patterns.

PCR-based sequence group typing

Two multiplex PCRs, designed to selectively amplify alleles of *ompA*, *csuE*, and bla_{OXA-51}-like genes with the specific primers listed in Supplementary Table 2, were used to assign the sequence groups and the corresponding major international clones I—III according to Turton et al. [3]. PCR conditions were as follows: denaturation at 94 °C for 3 min, followed by 30 cycles of 94 °C for 45 s, 57 °C for 45 s, 72 °C for 1 min, and a final extension at 72 °C for 5 min. Groups 1, 2, and 3 were identified according to Turton et al. [3].

Supplementary Table 2 related to this article can be found, in the online version, at http://dx.doi.org/10.1016/j.jiph.2015.04.030.

Statistical analysis

Because of the limited number of samples, the Fisher's Exact Test was employed to assess the correlation between antibiotic resistance and the relevant encoding genes. In order to estimate the *p*-value and its confidence interval, the Monte Carlo method was used. The data were analyzed using R software version 2.1.1.

Results

A. baumannii strains were recovered from burn ward. The origin of the isolates was wounds (30 out of 37; 81.1%), blood (6 out of 37; 16.2%), or urine

Table 1 Epidemiologic data and distribution of resistance phenotypes and genes among international clones of MDR and XDR *A. baumannii* strains.

Strain	Gender	Infection type	Source of isolation	MDR/XDR	*β-Lactamase* genes				Resistance or susceptible phenotypes					
					bla_{TEM}	bla_{PER}	bla_{OXA-23}	bla_{OXA-24}	IMP	MER	GM	TET	PTZ	AMS
1	Female	Wound infection	Wound swab	XDR	+	+	+	–	R	R	R	R	R	R
2	Male	Bloodstream infection	Blood culture	XDR	+	+	+	+	I	R	R	R	R	R
3	Female	Wound infection	Wound swab	MDR	+	+	+	+	R	I	R	R	R	S
4	Female	Wound infection	Wound swab	MDR	–	+	+	–	R	R	R	R	S	S
5	Male	Wound infection	Wound swab	XDR	–	+	+	+	R	R	R	R	R	I
6	Female	Wound infection	Wound swab	MDR	–	+	+	–	R	R	R	R	R	S
7	Female	Wound infection	Wound swab	XDR	–	+	+	–	R	R	R	R	R	R
8	Male	Bloodstream infection	Blood culture	MDR	–	+	+	+	R	R	R	R	R	S
9	Male	Wound infection	Wound swab	MDR	–	+	+	+	R	R	R	R	R	S
10	Male	Wound infection	Wound swab	MDR	–	+	+	–	R	R	R	S	R	S
11	Male	Wound infection	Wound swab	MDR	+	+	+	+	R	R	R	R	R	R
12	Male	Wound infection	Wound swab	MDR	+	+	+	–	R	R	S	R	R	R
13	Female	Wound infection	Wound swab	XDR	–	+	+	–	R	R	R	R	R	I
14	Female	Bloodstream infection	Blood culture	XDR	–	+	+	+	R	R	R	R	R	R
15	Female	Wound infection	Wound swab	XDR	–	+	+	+	R	R	R	R	R	I
16	Male	Bloodstream infection	Blood culture	XDR	–	+	+	–	R	R	R	R	R	R
17	Male	Wound infection	Wound swab	XDR	+	+	+	+	R	R	R	R	R	R
18	Male	Wound infection	Wound swab	XDR	+	+	+	–	R	R	R	R	R	R
19	Female	Bloodstream infection	Blood culture	XDR	–	+	+	+	R	R	R	R	R	R
20	Female	Bloodstream infection	Blood culture	MDR	+	+	+	–	R	R	R	R	R	S
21	Male	Wound infectionn	Wound swab	XDR	–	+	+	–	R	R	R	R	R	R
22	Female	Wound infection	Wound swab	MDR	+	+	+	+	R	R	R	R	R	R
23	Male	Wound infection	Wound swab	MDR	+	+	+	+	R	R	R	I	R	S
24	Female	Wound infection	Wound swab	MDR	+	+	+	–	R	R	R	R	R	S
25	Male	Wound infection	Wound swab	MDR	+	+	–	+	S	I	R	R	R	R
26	Female	Wound infection	Wound swab	MDR	+	+	–	+	S	I	R	R	R	R
27	Male	Wound infection	Wound swab	MDR	–	+	–	+	S	I	R	I	I	I
28	Male	Wound infection	Wound swab	MDR	–	+	–	+	S	R	R	R	R	R
29	Male	Wound infection	Wound swab	MDR	–	+	+	–	S	R	R	R	R	R
30	Female	Urinary tract infection	Urine culture	MDR	–	+	+	+	S	R	R	R	R	I
31	Female	Wound infection	Wound swab	MDR	–	–	–	–	S	S	R	R	S	S
32	Female	Wound infection	Wound swab	MDR	–	–	–	–	S	S	I	R	S	R
33	Male	Wound infection	Wound swab	MDR	–	–	–	–	S	S	R	S	S	R
34	Male	Wound infection	Wound swab	MDR	–	–	–	–	S	S	I	I	R	R
35	Male	Wound infection	Wound swab	MDR	–	–	–	–	S	S	S	I	R	R

Table 1 (*Continued*)

Strain	Gender	Infection type	Source of isolation	MDR/XDR	β-*Lactamase* genes				Resistance or susceptible phenotypes				
					bla_{TEM}	bla_{PER}	$bla_{OXA\text{-}23}$	$bla_{OXA\text{-}24}$	IMP	MER	GM	TET	PTZ	AMS
36	Male	Wound infection	Wound swab	MDR	—	—	—	—	S	S	R	I	S	R
37	Female	Wound infection	Wound swab	MDR	—	—	—	—	S	S	I	S	R	I

REP-PCR, repetitive element palindromic PCR; ICL, international clonal lineage; MDR, multidrug resistant; XDR, extensively drug resistant; IMP, imipenem; MER, meropenem; GM, gentamicin; CFE, cefepime; TET, tetracycline; PTZ, piperacillin–tazobactam; AMS, ampicillin-sulbactam; CIP, ciprofloxacin; POL, polymyxin B and E; R, resistance; S, susceptible; +, contained gene; –, gene not detected.

(1 out of 37; 2.7%) and were associated with wound infections, bloodstream infections, or urinary tract infection, respectively (Table 1).

Genotyping of *A. baumannii* isolates identified 11 different REP-PCR types that we labeled A through K, which showed a similarity of <80% in dendrogram analysis (Fig. 1). REP-PCR types I (11/37; 29.7%), E (8/37; 21.6%), B (3/37; 8.1%), and J (3/37; 8.1%) were the most frequently isolated (Fig. 1 and Table 1). The majority of isolates belonged to either international clone I (48.6%; 18 out of 37) or II (48.6%; 18 out of 37). Only one isolate belonged to international clone III (2.7%; 1 out of 37) (Fig. 1 and Table 1).

Antimicrobial susceptibility studies showed that 26 (70.3%) and 37 (29.7%) of the *A. baumannii* isolates were MDR and XDR, respectively (Table 1). None was resistant to colistin or polymyxin B (MIC ranges of both were 0.12–0.5 μg/mL; MIC_{50} 0.25 μg/mL and MIC_{90} 0.5 μg/mL). The highest resistance rate was observed against ciprofloxacin and cefepime (100%), followed by gentamicin (94.6%), piperacillin–tazobactam (86.5%), meropenem (81.1%), ampicillin-sulbactam (73%), and imipenem (64.9%). Sixteen of the 30 isolates (53.3%) resistant to meropenem and 13 of the 24 isolates (54.1%) resistant to imipenem belonged to sequence group 1, corresponding to international clone II. Correlations between antimicrobial susceptibilities, REP-PCR types, and international clones are shown in Table 2. Chi-squared and Fisher exact tests showed no significant correlation between imipenem and meropenem resistance and MDR, XDR, REP-types, and the presence of $bla_{OXA\text{-}24}$-like ($p > 0.05$); however, there was a significant difference between resistance to imipenem and meropenem and the presence of $bla_{OXA\text{-}23}$-like carbapenemase ($p \leq 0.05$).

All isolates harbored the $bla_{OXA\text{-}51}$-like beta-lactamase intrinsic to *A. baumannii* isolates. Eighty-one percent of cefepime-resistant isolates (30 out of 37) harbored the bla_{PER}-like gene (Table 1). The distribution of bla_{TEM}-like in cefepime-resistant isolates was 35.1% (13 out of 37) (Table 1). All imipenem- and meropenem-resistant isolates contained at least one $bla_{OXA\text{-}23}$ and/or $bla_{OXA\text{-}24}$-like carbapenemase. All imipenem-resistant and 86.7% of meropenem-resistant (26 out of 30) isolates harbored a $bla_{OXA\text{-}23}$-like gene flanked by *ISAba1* at the 5′end of the gene; 56.7% (17 out of 30) of meropenem-resistant and 50% (12 out of 24) of imipenem-resistant isolates contained the $bla_{OXA\text{-}24}$-like gene. The coexistence of $bla_{OXA\text{-}23}/bla_{OXA\text{-}24}$ was seen in 50% (12 out of 24) and 43.3% (13 out of 30) of imipenem-resistant and meropenem-resistant

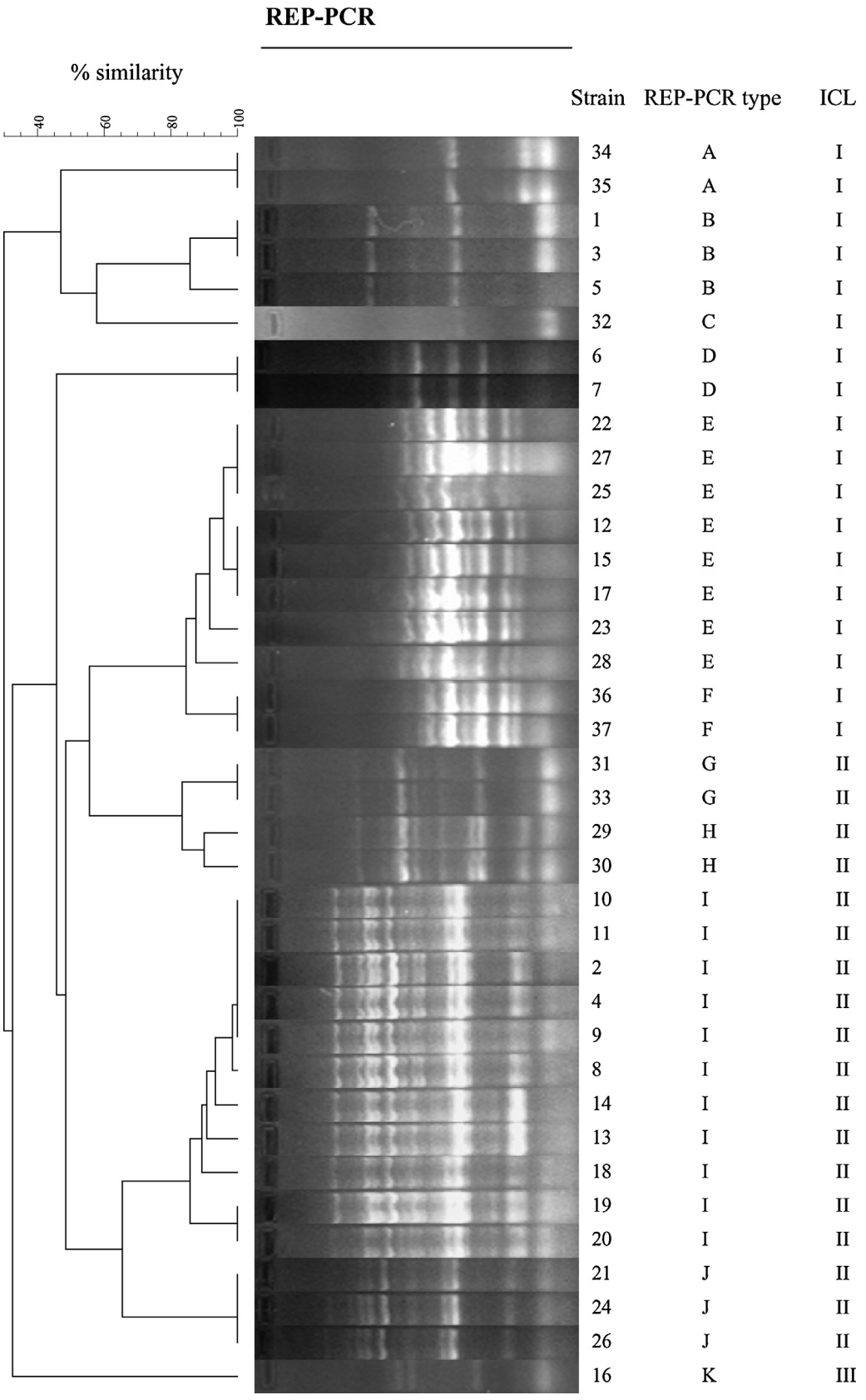

Figure 1 Cluster analysis of REP-PCR profiles of the *A. baumannii* strains isolated from burn patients. Strain number, REP-PCR profiles, and international clone assignments are indicated.

Table 2 Distribution of resistance phenotypes, genes and ICLs among *A. baumannii* isolates.

Carbapenem	Phenotype	*n* (%)	MDR *n* (%)	XDR *n* (%)	ICL *n* (%)			β-Lactamase resistant genes n (%)	
					I	II	III	$bla_{OXA\text{-}23}$	$bla_{OXA\text{-}24}$
Imipenem	Resistance	24 (64.9)	13 (54.2)	11 (45.8)	10 (41.7)	13 (54.2)	1 (4.2)	23 (95.8)	12 (50)
	Susceptible	13 (35.1)	13 (100)	0	8 (61.5)	5 (38.5)	0	2 (15.4)	3 (23.1)
Meropenem	Resistance	30 (81.1)	19 (63.3)	11 (36.7)	13 (43.3)	16 (53.3)	1 (3.3)	25 (83.3)	15 (50)
	Susceptible	7 (18.9)	7 (100%)	0	5 (71.4)	2 (28.6)	1 (2.7)	0	0

isolates, respectively. We could not detect any $bla_{OXA\text{-}58}$-like, $bla_{OXA\text{-}143}$, bla_{VEB}-like, bla_{IMP}-like, bla_{VIM}-like, bla_{SIM}-like, bla_{GIM}-like, or bla_{SHV}-like genes. ISA_{ba1}, upstream of and adjacent to $bla_{OXA\text{-}51}$-like and $bla_{OXA\text{-}23}$-like genes, was detected among all carbapenem-resistant isolates carrying these genes (30 out of 37; 81.1%). We did not detect IS_{1133} upstream of $bla_{OXA\text{-}51}$-like and $bla_{OXA\text{-}23}$-like genes.

Discussion

Although carbapenems, aminoglycosides, and fluoroquinolones have been used to treat *A. baumannii* infections in different medical settings, resistance to carbapenems has been increasing in Iran and in other countries [1,9—12]. The results of this study show that resistance to different antibiotics including carbapenems was high in *A. baumannii* isolated from burn patients in Tehran. In this study, only colistin and polymyxin B were active against all *A. baumannii* strains. This is concerning because these drugs have nephrotoxic and neurotoxic side effects [22,23]. Resistance rates to imipenem in Tehran hospitals increased alarmingly from 40.47% in 2008 to 48.9% in 2012 and to 59% in 2013 [9—11]. Trend analysis using the Chi-squared test showed that there is a statistically significant linear trend between resistances to imipenem in different years ($p \leq 0.03$). Concordantly, 64.9% of *A. baumannii* strains isolated from burn patients during 2011 described in the present study was resistant to imipenem. Resistance rates to imipenem and meropenem were 64.9% and 81.1%, respectively. In a previous study performed at this teaching hospital, resistance to imipenem was 57.3% [9], which was lower than that observed in the present study. Recent studies reported high rates of resistance to carbapenems in hospital settings in Tehran and Tabriz [10,11]. The increase in carbapenem resistance in *A. baumannii* strains isolated from burn patients in Iran can jeopardize treatment with carbapenems.

Mounting evidence indicates that production of carbapenemases belonging to class D β-lactamase is the most important mechanism for carbapenem resistance in *A. baumannii* isolates from Iran [8—11]. In accordance with previous studies [24—26], *A. baumannii* strains included in our study were resistant to imipenem or meropenem and contained $bla_{OXA\text{-}23}$-like, $bla_{OXA\text{-}24}$-like, $bla_{OXA\text{-}51}$-like genes, and/or combinations of these genes. Our data are also in agreement with those of previous studies showing that ISA_{ba1} provides the promoter required for expression of adjacent $bla_{OXA\text{-}51}$ and

bla$_{OXA\text{-}23}$ genes [24,25]. In fact, we also found *ISA*$_{ba1}$ upstream of and adjacent to *bla*$_{OXA\text{-}51}$-like and *bla*$_{OXA\text{-}23}$-like genes in all carbapenem-resistant isolates carrying these genes. Other β-lactams such as metallo-β-lactamase *bla*$_{IMP}$ and *bla*$_{VIM}$ have been reported sporadically in *A. baumannii* isolates from Iran [8] and from Tabriz province [11], but not from the *A. baumannii* isolates from Tehran described herein and in a previous study [9]. Based on the data shown herein, we can postulate that carbapenem resistance in *A. baumannii* strains isolated from burn patients in Tehran was selected by the spread of *bla*$_{OXA\text{-}23}$-like, *bla*$_{OXA\text{-}24}$-like, or *bla*$_{OXA\text{-}51}$-like genes. Our data identified a restricted number of REP-PCR genotypes with REP-PCR types E and I occurring in *A. baumannii* strains isolated from 8 and 11 patients, respectively. Owing to the lack of sufficient molecular epidemiology data regarding clonal circulation of drug-resistant *A. baumannii* in this hospital, we cannot definitively comment on the clonal circulation of MDR and XDR *A. baumannii*; however, according to the results of this study and those of Bahador et al., it seems that the spread of MDR and XDR *A. baumannii* strains among burn patients in Iran may be mostly due to cross-transmission of a few predominant genotypes between patients. Identification of three concurrent MDR international clonal lineages (I—III) among *A. baumannii* strains isolated from burn patients in Tehran is noteworthy. These resistant clones were first reported from European hospitals. However, they quickly spread to other hospitals worldwide and were then considered international clones [2]. The incidence of MDR *A. baumannii* is increasing worldwide; carbapenem could become an important selector for MDR and XDR *A. baumannii*. Therapeutic options for treating infections with these bacteria are becoming increasingly limited. [27,28].

Our data show that international clones 1 and II, producing *bla*$_{OXA\text{-}23}$-like and *bla*$_{OXA\text{-}24}$-like carbapenemases, are predominant among the *A. baumannii* strains isolated from burn patients in Tehran [10]. The spread of carbapenem-resistant *A. baumannii* clonal lineages I and II producing *bla*$_{OXA\text{-}23}$-like and *bla*$_{OXA\text{-}24}$-like carbapenemases has been observed in Europe, Asia, and Latin America [2,29,30]. Our data are in agreement with those of two studies from Tehran and Tabriz reporting the spread of international clones I and II in Iran [10,11]. In the present study, one strain belonging to international clone III was isolated for the first time from burn wound infections. Interestingly, *A. baumannii* clonal lineages I and II isolated from burn wounds of burn patients are identical to those isolated from hospitalized patients in other clinical settings and from other sources [2,29—31]. The isolate belonging to international clone III was MDR and harbored genes for *bla*$_{OXA\text{-}51}$-like, *bla*$_{OXA\text{-}23}$-like + *IS*$_{Aba1}$, *bla*$_{PER}$-*like*, and the *adeABC* efflux pump. Unlike other reports from Iran, European countries, Asia, and Latin America [2,3,9—11,17,28,29], we did not detect *bla*$_{OXA\text{-}58}$-like carbapenemase in our isolates. Thus far, there are a few reports are available about microorganisms that are the most frequently isolated from burn patients in Tehran. Although Tehran has more than 130 hospitals, only a few specialize in the treatment of burns, and among these, the Motahari Burn and Reconstruction Center is the largest, and its importance and relevance to the treatment of burns cannot be overstated. A glance at previous studies by us and other researchers in Tehran shows that gram-negative bacilli such *P. aeruginosa*, *Klebsiella pneumoniae*, and *A. baumannii* are frequently isolated from clinical specimens, especially from wounds, blood, and tracheae, and some genes play important roles in the antimicrobial resistance developed to the antibiotics used. In these studies, *bla*$_{OXA}$ carbapenemase, *bla*$_{VIM}$, *bla*$_{IMP}$, and *bla*$_{KPC}$ were introduced as major resistance genes encoding resistance to carbapenems [8—11,32—35]. Owing to the status of burn patients who are at high risk for skin infection and septicemia, most of the bacteria were isolated from wounds and/or blood. However, from some patients who were hospitalized in ICU wards, tracheal samples were taken because of the occurrence of ventilator-associated pneumonia [8—11,32—35].

Conclusion

Our data show that *A. baumannii* infections in burn patients in Iran were caused by the spread of two MDR and carbapenem-resistant epidemic clonal lineages, which were assigned to international clonal lineages I and II. The presence of *bla*$_{OXA\text{-}23}$-like and/or *bla*$_{OXA\text{-}24}$-like carbapenemases might have contributed to the establishment of the epidemic clones among burn patients. The increase in infections caused by carbapenem-resistant *A. baumannii* in burn patients suggests the necessity for a surveillance program that involves monitoring of health care-associated infections, molecular typing of microbial isolates, and characterization of antimicrobial resistance.

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Competing interests

None declared.

Ethical approval

Not required.

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